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From: Jagoe, Donna
Sent: Wednesday, September 03, 2003 11:56 AM
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Subject: bipolar vs anticonvulsant

KEY ABSTRACTS:

1: J Affect Disord 2002 May;69(1-3):1-14

Synaptic, intracellular, and neuroprotective mechanisms of anticonvulsants: are they relevant for the treatment and course of bipolar disorders?

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Treatment of bipolar disorders has progressed significantly in the last decade due to advances in basic and clinical research. Much of this progress has centered on the development of a new generation of mood stabilizers-anticonvulsants. Valproic acid (VPA) and carbamazepine (CBZ) have clear mood stabilizing properties, while lamotrigine (LTG), topiramate (TPM), and gabapentin (GBP) have been investigated to varying degrees. We provide an overview of mechanisms of these potentially mood-stabilizing anticonvulsants, review their commonalities and dissociations to the gold standard non-anticonvulsant mood stabilizer lithium. Regulations of the glutamate excitatory neurotransmission and/or gamma aminobutyric acid (GABA) inhibitory neurotransmission are mostly studied mechanisms of anticonvulsants. The divergent effects of these agents indicate that this mode of action represents initial effect of anticonvulsants in regulating mood. Similar to lithium, intracellular mechanisms of anticonvulsants, primarily VPA and CBZ, include regulation of several protein kinase signaling pathways, leading to regulation of gene expression. Common genes that can be regulated by mood stabilizers are more likely to be the final normalizing components in bipolar disorders. Several anticonvulsants, such as VPA, LTG, and TPM, show neuronal protective function, a commonality with recently identified neuroprotective function of lithium, although the meaning of neuroprotection in bipolar disorders remains to be identified. Understanding the mechanisms of anticonvulsant mood stabilizers, integrated with clinical observations, may ultimately provide important new insights into the pathophysiology and treatment of bipolar disorders.

PMID: 12103447 [PubMed - in process]

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